Loyola University Chicago

Loyola eCommons

Dissertations   Theses and Dissertations

1987

A Neuropsychological Investigation of Prefrontal Dysfunction among Conduct Disordered Adolescents

Robin Shear
Loyola University Chicago

Recommended Citation

Shear, Robin, "A Neuropsychological Investigation of Prefrontal Dysfunction among Conduct Disordered Adolescents" (1987). *Dissertations*. 2532.
https://ecommons.luc.edu/luc_diss/2532

A NEUROPSYCHOLOGICAL INVESTIGATION OF

PREFRONTAL DYSFUNCTION AMONG

CONDUCT DISORDERED ADOLESCENTS

by

Robin C. Shear

A Dissertation Submitted to the Faculty of the Graduate

School of Loyola University of Chicago in Partial

Fulfillment of the Requirements for the Degree of

Doctor of Philosophy

June

1987

ACKNOWLEDGEMENTS

VITA

Robin C. Shear was born in Muncie, Indiana on April 13, 1950, the son of Robert Clayton Shear and Lois (Siberry) Shear.

His elementary and secondary education were at Burris Laboratory School, Ball State University, Muncie, Indiana, where he graduated as salutatorian and Senior Class President in 1968. Mr. Shear was subsequently awarded an academic scholarship to Dartmouth College, Hanover, New Hampshire. In 1972, he received his baccalaureate Magna Cum Laude, with high departmental distinction in his major field of Visual Studies. During his Senior year at Dartmouth, Mr. Shear was designated a Rufus Choate Scholar, and won the Marcus Heiman Award for his contributions in the arts.

He pursued his interest in the arts at Washington State University, Pullman, Washington, receiving a Master of Fine Arts degree in 1974.

Mr. Shear then embarked on a six year career as a teacher in private secondary schools, most notably serving as Chairman of the Fine Arts Department of Texas Military Institute, San Antonio, Texas, from 1976 through 1981. He has been a member of the National Art Education Associa-

tion and the San Antonio Artist's Alliance. His paintings and drawings have been exhibited in both regional and national juried exhibitions.

In September, 1981, Mr. Shear entered Loyola University's doctoral program in Clinical Psychology, after first completing two years of preparatory graduate work in Clinical Psychology at Trinity University in San Antonio, Texas. He completed an APA approved Clinical Psychology Internship at Wilford Hall United States Air Force Medical Center, San Antonio Texas, in 1984, while also serving as an active duty Air Force Officer, with rank of Captain. Upon completion of this internship, he was assigned to the United States Air Force Hospital at Grissom Air Force Base, Indiana, where he was Chief of Mental Health Services. Since completing his Air Force tour in 1986, he has worked on the Psychiatric Inpatient units of Tipton Memorial Hospital, Tipton, Indiana, and Howard Community Hospital, Kokomo, Indiana.

TABLE OF CONTENTS

LIST OF TABLES

INTRODUCTION

Children and adolescents who exhibit persistent and
repetitive patterns of antisocial behavior, yet appear
otherwise normal, can be given the psychiatric diagnosis
of conduct disorder according to the third edition of the
Diagnostic and Statistical Manual of the American Psychi-
atric Association (1980). Conduct disorders are defined
not so much by any specific behaviors or qualities, as by
the disruptive, destructive, or obstructive effects they
have upon a larger group or institution. Because of these
deleterious effects upon others, conduct disorders consti-
tute a problem which has ramifications not only for the
mental health community, but for society as a whole.

The serious nature of this problem is evidenced by
the notably high prevalence, poor prognosis, and pessimis-
tic treatment outlook afforded conduct disorders. Although
there are no precise estimates of prevalency, some general
population surveys suggest that "conduct problems serious
enough to alarm some adult" occur among 5 to 15 percent of
all children (Meeks, 1980). The most comprehensive
prognostic study suggests that a high proportion of
antisocial children continue to exhibit antisocial behav-
ior into adulthood, and also appear to be at risk for

1

a variety of life-long adjustment problems (Robins, 1966). Furthermore, adults who were identified as antisocial during childhood also appear to have "more marital difficulties, poorer work records, worse social relationships, more psychiatric disorders and, to some extent, even poorer physical health" than do those who were not antisocial as children (Rutter, 1970).

The poor prognosis for conduct disorders has persisted despite efforts to apply psychiatric and psychologically oriented interventions. Both biological and psychotherapeutic interventions have been inconsistently effective, at best (Tucker & Pincus, 1980). The lack of treatment success has been so pronounced that, for many professionals, the term "antisocial behavior" has become a criteria for denial of treatment (Lewis & Balla, 1976). Further evidence of pessimism is found in the suggestion of some authors that the most effective treatment may simply be isolation from society until middle age, since antisocial behavior appears to decline in frequency after the age of 40 (Pincus & Tucker, 1978).

What accounts for the lack of treatment success with conduct disorders? One possibility is that the diagnosis of conduct disorder may simply be too general, lacking sufficient specificity for treatment to be effectively applied. Within the diagnostic category may be

several distinct subgroups, varying in etiology and in response to treatment, a situation which would enfeeble any single mode of treatment directed at conduct disorders as a whole. A crude analogy might be drawn between such a situation and an attempt to treat all fevers with a single antibiotic. Some of the patients, of course, would improve, but many would remain untouched because the diagnosis of fever was not specific enough to determine appropriate treatment.

Notably, most of the research relevant to the treatment of conduct disorders has assumed the conduct disorders to be a unitary entity. Research has typically been conducted using groups exhibiting antisocial behavior defined in global terms, without reference to possible qualitative differences within the groups.

Consider, for example, the following criteria used to define groups of antisocial individuals in research studies: general disrespect and defiance of school rules, (e.g., stealing, fighting and/or truancy), resulting in frequent minor punishments, detentions, and/or temorary suspensions (Saklofske, McKerracher, & Eysenck, 1978); classroom disturbance, disrespect and defiance (Saklofske, 1977); adjudication for delinquency (Peterson, Quay, & Cameron, 1959); social disapproval in classrooms, disruptive and aggressive behaviors (Feldhusen, Benning, &

Thurston, 1972); placement as inmates in a training school
(Peterson, Quay, & Anderson, 1959).

Note that the above criteria, for the most part,
refer to the effects or consequences of antisocial behav-
ior, ignoring both the great variety of specific behavior
which can produce such effects and the variety of cogni-
tive, physiological, emotional and motivational variables
affecting the individuals who produce the behavior. Such
variations may, in fact, constitute differences crucial to
differential diagnosis, and consequently to effective
application of treatment. If this is the case, then
treatment effectiveness could be improved by more specific
diagnoses - by delineating subgroups within the conduct
disorders. Only then could an effective range of treat-
ment be developed, varying with the significant quali-
tative differences among behavior and individuals.

Where does one begin in the attempt to delineate
diagnostically important subgroups within the conduct
disorders? Although there are undoubtedly many possible
starting points, this researcher has been led, by indepen-
dent clinical observation of conduct disordered adoles-
cents, to question whether some of these individuals
suffer from a reduced ability to alter their behavior in
response to changing circumstances. A disturbance of
behavior control of this type can also be observed in

patients with known pathology of the frontal lobes of the cerebrum. These observations are consistent with the speculation of some researchers that one subgroup within the conduct disorders can be defined in terms of symptoms of frontal lobe dysfunction, possibly developmental in origin (Pontius, 1972; 1973). This subgroup would be seen to exhibit behaviors qualitatively resembling those of frontal lobe impaired individuals and qualitatively distinct from those of other conduct disordered individuals.

Identification of this subgroup would be a first step toward developing differential diagnoses within the conduct disorders, and eventually more specific treatment. The study presented in this paper attempts to investigate the validity of conceptualizing a subgroup of the conduct disorders in terms of frontal lobe impairment. The general strategy for doing so is to first identify a subgroup exhibiting behaviors qualitatively resembling those associated with frontal lobe impairment, and then to determine whether the subgroup also exhibits neuropsychological deficits consistent with frontal lobe impairment. Convergence between the behaviors used to identify the subgroup and the neuropsychological measures can then be viewed as bolstering the predictive validity of the conceptualization.

LITERATURE REVIEW

The body of literature directly investigating the relationship between neuropsychological measures of frontal lobe dysfunction and conduct disorders is limited. The more general notion of causal relationship between organic factors and antisocial behavior, however, has deep historical roots. It will be helpful to first summarily review these in that they provide a conceptual context for the current study. Secondarily, an overview of the nature and symptomatology of frontal lobe dysfunction will be provided. This will be followed by a description of the way conduct disorders might result from such dysfunction in some individuals, and how such dysfunction might be measured. Finally the literature directly investigating the relationship between neuropsychological measures of frontal lobe dysfunction and conduct disorders will be reviewed, with the intent of ascertaining the degree to which such relationship has been clarified.

Historical Context

One of the earliest conceptualizations of the role of organic factors in behavior was that of the early Greeks, who viewed the personality as emerging from the interaction of four bodily fluids or "humours". Antiso-

cial as well as other abnormal behavior was seen as resulting from a deviant or imbalanced mix of the humours. Variations of this view of antisocial behavior as springing from a general, but rather non-specific physiological substrate were held by as recent an authority as Lombroso (1910), who considered criminals and delinquents to be "constitutional deviants", in some way fundamentally (i.e., organically) different from normal human beings. The idea that problems of conduct and behavior might result from dysfunction of specific brain regions and/or structures emerged only with the advent of the case study method in the nineteenth century, and more specifically from case studies of head-injured individuals.

One of these nineteenth-century cases, so famous that it is cited in many abnormal psychology textbooks, provides early evidence of a link between frontal lobe dysfunction (at least of the gross sort caused by direct and substantial trauma) and disturbance of social conduct and impulse control. Phineas Gage, an apparently responsible and reliable railroad foreman prior to an accident in which the frontal aspect of his skull was pierced by a steel rod, subsequently developed what can only be described as "antisocial" personality characteristics. Despite all evidence of physical recovery following the accident, Gage was observed be "fitful, irreverent,

indulging at times in the grossest profanity (which was not previously his custom), manifesting but little deference for his fellows, impatient of restraint or advice when it conflicts with his desires, at times pertinaciously obstinate, yet capricious and vacillating, devising many plans of future operations, which are no sooner arranged than they are abandoned in turn for others..." (Coleman, Butcher & Carson 1980, 451-452).

The twentieth century has seen the emergence of the idea that antisocial behavior, especially among children, may also result from brain dysfunction of a lesser degree and/or of less obvious origin than that experienced by the unfortunate Mr. Gage. This notion probably originated in the observations made of child victims of the 1917-1918 lethargic encephalitis epidemic, who were seen to commonly develop symptoms of hyperactivity, antisocial behavior and emotional instability despite apparent physical recovery (Rutter, 1982; Werry, 1979). It appears to have awaited formal expression until the 1940's, when the concept of the "minimally brain damaged child" appeared in the literature (Gesell & Armatrauda, 1941; Strauss & Lehtinen, 1947).

In its early form, the concept of minimal brain damage held that a characteristic cognitive and behavioral syndrome, which included hyperactivity, impulsivity,

emotionality, and learning deficits, was associated with lesser degrees of brain damage in children, regardless of location and etiology of the damage. As in the old "constitutional deviance" theory, behavioral problems were viewed as resulting from a unitary, continuous variable, having non-specific effects, the nature and severity of which depended primarily upon the quantity of dysfunctional brain tissue, rather than upon the location or etiology of the damage. In cases where no obvious history of trauma or physiological problems could be observed, the presence of the syndrome could be taken as indicative of underlying brain damage (Werry, 1979).

This early concept was refined in the 1950's and 60's by Pasamanick and Knobloch (1960) who hypothesized, in their studies of outcomes of pregnancy complications, that the effects of prenatal and birth process brain damage varied along a "continuum of reproductive causalty". With severe damage, recognizable neurological disorders developed; when the damage was mild, there was a tendency for behavioral difficulties, unaccompanied by overt neurological abnormality, to occur.

The "non-specific" version of the minimal brain damage (MBD) hypothesis was and remains highly influential. By the 1970's, however, there appeared to be

sufficient reason to doubt its validity (Werry, 1979).
Its problematic aspects are summarized below.

First, the range of symptoms attributed to MBD was
too broad to constitute a single, well defined syndrome.
In a thorough review of the literature, Clements (1966)
found 99 separate symptoms referred to as resulting from
MBD. Even the 10 most frequently cited of these symptoms
forms a rather nebulous array: 1) hyperactivity; 2)
perceptual motor impairments; 3) emotional lability; 4)
general coordination deficits; 5) disorders of attention
(short attention span, distractibility, perseveration); 6)
impulsivity; 7) disorders of memory and thinking; 8)
specific learning disabilities; 9) disorders of speech and
hearing; 10) equivocal neurological signs and electroen-
cephalographic irregularities (Clements, 1966). The range
of symptomatology ascribed to MBD not only made research
difficult: it also cast doubt on the clinical utility of
the concept. As one clinician put it: "the (symptoms)
seen as a result of brain damage are in fact so diverse
that it is doubtful whether the concept (of the brain
damaged child) has any useful validity at all, except
perhaps as a piece of convenient clinical shorthand to
refer to a great group of disturbances that appear in some
way to be different from the general round of psychologi-
cal disorders in childhood" (Pond, 1967).

Second, multivariate statistical research investigating possible relationships within the diversity of symptomatology ascribed to MBD found no evidence of groupings or subgroupings suggestive of a cohesive syndrome (e.g., Jenkins, 1964; Schulman, Kaspar, & Throne, 1965). This was true even when consideration was limited only to Clements' (1966) 10 most frequently cited symptoms of MBD (Routh & Roberts, 1972).

Third, as numerous British researchers have pointed out, the non-specific view of the effects of brain damage is incompatible with well known data concerning localization of function in the brain (McFie, 1975; Rutter, Graham, & Yule, 1970), which suggests that the specific effects or symptoms produced by brain damage vary with the site of the lesion and the age at which it occurs.

Finally, there was a persistent lack of evidence pointing to a connection between the symptoms of concern in MBD, and "hard" brain damage (Rutter, 1982). Chess (1972), for example, in an extensive retrospective study of children encountered in her clinical practice, found that of the symptoms commonly thought to be associated with brain damage, only perseveration was statistically characteristic of those children with known brain damage (i.e., those with observable neurological symptoms). Furthermore, it had become evident that the symptoms of

concern could often be viewed as developmental rather than abnormal in nature. In order to accomodate this lack of evidence the "minimal brain damage" concept was "softened" to that of "minimal brain dysfunction", evidenced primarily by impaired performance on neuropsychological measures and by neurological "soft" signs (Werry, 1979). For the remainder of this paper, the letters "MBD" will symbolize this latter term.

The above problems generated a rethinking and modification of the MBD concept. Many researchers felt the general concept of MBD was sound, and tried to preserve a semblance of the "non-specific" version while accomodating the issues raised by others. Wender and Eisenberg (1974), for example, in their summary article, acknowledge on the one hand that "children so affected (by MBD) differ markedly from one another, presumably in relationship to the presence or absence of an anatomical lesion, size of the lesion, site of the lesion, number of lesions, the age of acquisitions, the total amount of brain tissue involved, and perhaps even the cause of the lesions," yet insist there is "sufficient commonality to the behavioral syndromes and sufficient responsiveness to similar treatment regimes to warrant the continued clinical use of the diagnostic term" (page 131).

For most authors, however, phenotypic resemblances among behaviors and responses to treatment did not constitute grounds for assuming a common genotype, or unitary etiology. In general, as Werry (1979) notes, there has been a "clear movement away from the simplistic (i.e., non-specific) notion of minimal brain dysfunction" (page 111), and toward the more complex view of multiple distinct subgroups within the classification. The basic research problem implied by this more complex view is one of differentiating among possible subgroups. A rather comprehensive set of criteria for defining subgroups, and thus for guiding research efforts, has been suggested by Clements (1966): 1) by symptoms grouped on the basis of localization of brain dysfunction; 2) by empirically derived symptom clusters; 3) by psychophysiological response patterns; 4) by presence of minor physical anomalies; 5) by response to medication; 6) by biochemical studies. There have been significant efforts along each of these lines, and while it is beyond the scope and intent of this paper to review the work that has been accomplished along each, the interested reader is referred to Werry (1979) for specific citations.

The current study clearly fits into the context of these efforts in that it follows the first of Clements' suggestions: that of defining a subgroup on the basis of

symptoms associated with localized brain dysfunction. That other efforts following this suggestion have been fruitful and well accepted is evidenced by the inclusion of specific developmental disabilities as diagnoses in the third edition of the American Psychiatric Association's Diagnostic and Statistical Manual (1980). Such disabilities are thought to result from dysfunction of specific cortical areas (e.g., receptive language disabilities are thought to be correlated with the temporal region of the left hemisphere).

The idea that a specific developmental disability might similarly result from frontal lobe dysfunction caused by a "fixation at the phase of normal immaturity, or a maturational lag, or some as yet unknown pathology of the frontal lobes and/or the caudate nucleus" appeared as early as the 1970's, notably in the work of Pontius (1973, p. 61). Individuals with such a disability might be recognizable by their presentation of symptoms and signs consistently found in frontal lobe dysfunction. Furthermore, as there were parallels between these symptoms and behaviors of some types of delinquents, it was speculated that there might be a causative relationship between frontal lobe disability and a proportion of conduct problems. In order to understand how such a relationship

could occur, it will be helpful to first consider the
symptomatology associated with frontal lobe dysfunction.

Frontal Lobe Dysfunction

The frontal lobes consist of all of the tissue
forward of the central sulcus. In general, they comprise
the brain's motor system, and as such are thought to be
involved in the control and regulation of behavior. The
regions closest to the central sulcus have specific roles
in the control of movement, and lesions to these areas can
produce severe, chronic, and obvious deficits in fine and
gross motor control, speed, strength and coordination
(Kolb & Whishaw, 1980). It is the portion of the brain
further forward from the central sulcus, however, the
prefrontal cortex, that is of greatest interest to the
problem at hand.

The prefrontal cortex is the site of a dense
network of interconnections with both the limbic system
and posterior cortex. These presumably supply input from
other brain structures which modifies or regulates move-
ment or behavior, and also provide feedback to the rest of
the brain regarding the ongoing behavior. Thus, the
prefrontal cortex is where "already correlated incoming
information from all sources - external and internal,
conscious and unconscious, memory storage and visceral
arousal centers - is integrated and enters ongoing

activity" (Lezak, 1983). In contrast to the specific role played in movement by the portions of the frontal lobes nearer the central sulcus, the prefrontal cortex appears to have a "nonspecific role in movement control, and probably plays little role in the actual control of the components of movement. Rather, the prefrontal cortex controls the overall motor programs and adds flexibility to motor output by modifying behavior with respect to specific internal and external factors" (Kolb & Whishaw, 1980). In all, the role of the prefrontal cortex is that of adapting and adjusting - "fine-tuning" behavior to appropriately fit changing circumstances.

As might be expected, impairment of the prefrontal region, rather than producing observable effects upon movements themselves, appears to disrupt feedback among ongoing behaviors and information provided by other brain structures regarding the internal states and external situations of the organism. The "reciprocal relationships between the major functional systems - the sensory system of the posterior cortex, the limbic-memory system with its interconnections to subcortical regions involved in arousal, affective, and motivational states, and the effector mechanisms of the motor system" may all be damaged (Lezak, 1983). As a result, behavior generally becomes inflexible, and fails to be easily affected by its

consequences or by changing circumstances. "The effect of action is not evaluated, no signal of errors is actual-ized, mistakes are not corrected" (Luria & Homskaya, 1964).

This inflexibility is such that there is diffi-culty carrying out a complex series of actions when steps in the chain of actions require arrest or alteration of the preceding action. There is a tendency, instead, to in some way continue with the ongoing step. The following three illustrations from Luria's work will help clarify this.

#1. A severe case: "a patient with massive tumor of the frontal lobes is asked to light a cigarette...even such a simple action which includes several successive links proves to be impossible; the patient begins to strike a match, and continues many times to strike it, unable to shift to the next action required to light the cigarette" (Luria & Homskaya, 1964, p. 358).

#2. A patient with less severe damage is able to carry a simple instruction to light a cigarette, "but if the instruction is more complicated, if, for example, the patient is asked to light a candle, the task becomes impossible. The whole pattern of the action disinte-grates, the patient begins by lighting the match and then blows it out, or he puts the candle in his mouth,

reproducing the act of smoking. Verbal programming of a complex action is disturbed by strong, stable fragments of former programs; no matching of the effect of action with the instruction is accomplished, and no evaluation of results and correction of errors follows" (p. 358).

#3. A similar difficulty in arresting an ongoing behavior in response to a environmental demand for different behavior can be observed in young children, prior to the age - 3 1/2 to 4 years - at which the frontal lobes develop the ability to function effectively. " If an 18-month-old child who has started to put rings on a stick receives a verbal instruction to take the rings off, he continues to put the rings on the stick, and even accelerates this action, being unable to arrest the action he has begun and shift to the opposite behavior required by the verbal instruction" (p. 357).

This particular kind of inflexibilty, marked by the failure of ongoing behavior to readily shift in order to accomodate changing internal and external circumstances can be seen as an integral, if not the fundamental (Milner, 1964) characteristic of all symptoms that are generally associated with prefrontal impairment. The following consideration of the five general groups of behavioral disturbances associated with frontal lobe

damage as suggested by Lezak (1983, p. 81-82) should clarify this point.

The first of these groups is problems of starting: decreased spontaneity, decreased productivity, decreased rate at which behavior is emitted, or decreased or lost initiative...severe problems of starting appear as apathy, unresponsiveness, or mutism." This group may also be thought of as a difficulty in shifting from an ongoing state of inertia, or absence of behavior in response to a demand for increased production. A frontal impaired individual may find it difficult to initiate behavior or production simply because to do so requires an initial modicum of flexibility.

The second is difficulties in making behavioral or mental shifts: "shifts in attention, changes in movement, or flexibility in attitude." Such difficulties occur supramodally, that is, across a variety of situations and tasks. They often appear as a type of perseveration, as "difficulty in suppressing ongoing activities or attention to prior stimulation. On intellectual tasks, it may be expressed in repetitive and uncritical perpetuation of a response that was once correct but becomes an uncorrected error under changed circumstances or in continuation of a response beyond its proper end point." This group consti-tutes the obvious case of failure to readily shift.

Problems in stopping are the third group. These "show up in impulsivity, overreactivity, disinhibition, and difficulties in holding back a wrong or unwanted response, particularly when it may either have a strong association value or be part of an already ongoing re-sponse chain." This group may be thought of similarly to the first group: there is difficulty shifting from the ongoing behavior when circumstances are changed.

Deficient self-awareness - "an inability to per-ceive performance errors, to appreciate the impact one makes on others...", and a concrete attitude, with which "the patient becomes incapable of planning and foresight or of sustaining goal directed behavior" are the other two groups. These symptoms can be viewed as inferentially derived from observed failures to shift behavior in response to changing circumstances. If, for example, an individual persistently fails to shift an ongoing behavior despite negative social consequences, then one might infer that the individual was insufficiently aware of his effect upon others. All that has been directly observed, howev-er, is the failure to readily make such a shift.

While behavioral disturbances like the above tend to be supramodal, that is, tend to occur across a variety of situations and tasks, there is also some evidence for localization of specific function within the prefrontal

area. Left hemisphere lesions, for example, produce more
interference with control of language behavior, while
right hemispheric lesions produce greater noverbal defi-
cits (Jones-Gotman & Milner, 1977; Kolb & Milner, 1981;
Kolb & Whishaw, 1980). It also appears that lesions in
the dorsolateral (upper) portion of the prefrontal cortex
have greatest impact upon cognitive phenomena while
lesions in the orbitomedial (lower) area have a more
specific effect upon emotional and social behavior (Lezak,
1983; Milner, 1963). What is noteworthy, however, is that
the manner in which lesions affect these various modes of
behavior is similar, with an integral component being some
form of difficulty in making shifts (whether cognitive,
behavioral, linguistic, or emotional) in response to
changing internal and external demands.

Frontal Lobe Dysfunction and Conduct Disorders

How might such impairment lead to a diagnosis of
conduct disorder? Why would some individuals with this
kind of impairment be found among juvenile delinquents?
Clearly, some of the symptoms associated with frontal lobe
impairment sound similar to traits commonly ascribed to
antisocial individuals: lack of foresight, impulsiveness,
lack of appreciation for one's impact on others, etc.
However, if difficulty in making shifts as described above
is integral to frontal lobe impairment, then one would

expect this difficulty to be central to the development of conduct problems in some adolescents. This is precisely what Pontius (1972) suggests.

Consider a child with such a disability who enters a toy store, for example, and, perhaps as instructed by a parent, begins to carefully handle some of the toys in order better examine them. "Suddenly the storekeeper approaches, shouting not to touch the toys. Such a child may not be capable of reprogramming his action on verbal command; of switching from the plan and principle guiding his ongoing action to a new plan of action with a new overriding value. He continues his principle of carefully handling the toy - one which he just happened to have picked up at the moment the verbal command reached him. He leaves the store, toy in hand, having been triggered by the gestures of the storekeeper, but not reprogrammed by his verbal command. He knows all through this behavior, that it is wrong to "steal" and he has no such conscious or unconscious intentions. Afterwards he may feel genu- inely guilty and especially upset about what he has done. When asked, he says he feels he is a `bad boy,' that everybody has told him so, that he has done something bad again...He is puzzled and at a loss, and indeed he may well have suffered a neurologically based loss of mastery over his actions" (Pontius, 1972, p. 294).

A child behaving in the above way may also have elicited unjustified reproach or punishment. He may feel defeated and resentful, and may express himself through negative or aggressive behavior, which in turn may lead to further reproach and punishment. With further similar incidents, it is easy to see how a vicious cycle might develop, leading to an adversarial relationship with authority, an antisocial attitude, and a "delinquent" or "bad boy" self image. This child might then become attracted to and involved with other "bad" individuals, and participate in the activities of this peer group. Eventually, his disability might lead to even further difficulties.

With a group of friends, he breaks into a house. "He knows well that hurting a person is much worse ethically speaking than stealing, and he has no intention of going beyond stealing...As he is in the house,...the owner appears and shouts at him to stop. This sudden new stimulus calls for flexibility, for reprogramming his principle of action, his values...As he continues to follow his initial principle of action to get the money, he just eliminates any obstacle in his way. Thus he may grab a nearby object, hit the owner with it, and perhaps even kill him...under these changed external circumstances (into which pressure and emotional response also enter,

(aside from verbal interaction) he is unable to reprogram his ongoing activity" (p. 294). According to Pontius, it is precisely this inability to reprogram ongoing behavior, to make "shifts" in response to changing circumstances, which would distinguish a frontal lobe impaired subgroup from the larger group of antisocial or conduct disordered children.

Measures of Frontal Lobe Dysfunction

Given that the specific difficulty of making mental or behavioral shifts in response to changing environmental circumstances may be observable in the antisocial behavior of some individuals, and , may distinguish frontal or prefrontal impaired individuals from others with conduct problems, the question arises as to whether cognitive or neuropsychological measures might be sensitive to these same difficulties. There are a number of tests generally associated with frontal lobe functioning in the clinical literature (e.g., Lezak, 1983; Kolb & Whishaw, 1980). There are, however, no studies comparing their relative discriminatory capabilities, and so a degree of arbitrariness necessarily accompanies the preferential use of any particular test. For the purposes of the current study, the following rationale was used to select an appropriate set of measures.

First, each measure had to have good face validity. It had to reasonably and arguably consist of a task requiring mental or behavioral shifts in accordance internal and/or environmental circumstances. In this way it would presumably be sensitive to the effects of prefrontal impairment.

Second, there had to be clinical or experimental documentation as to the sensitivity of the measure to frontal-lobe impairment. There was an attempt to find measures that were sensitive exclusively to frontal lobe impairment, but as with most neuropsychological measures, impaired performance on a given task can also often result from dysfunction in other brain areas (e.g., any task involving visual perception and/or processing will be affected by occipital dysfunction).

Furthermore, there is considerable variation in the amount of research, replication, and standardization that has gone into the development of each test, and therefore some questions regarding differential effects of lesion type and site, and of other variables remain unaddressed. As much as possible, the selected tests had to be backed by documentation suggesting specific sensitivity to frontal lobe impairment. If impairment elsewhere in the brain also affected test performance, this effect had to be less pronounced than the frontal effect. Since the

current study involves comparison between two experimental groups, and any relative differences between the groups are of primary concern, it was not important that all tests be standardized or normalized.

Finally, within the set of measures selected, there had to be as wide a variety of specific task as possible, so as to demonstrate supramodality of dysfunction, should it occur. In this regard, tasks respectively emphasizing language and non-verbal abilities (i.e., dominant and non-dominant hemispheres) were included, as were tasks involving various levels of cognitive functioning.

The tests described below are those selected for the current study. The descriptions touch upon each of the above points.

Speech Fluency Task. There are several versions of this task (Lezak, 1983), including written versions based on the Thurstone Word Fluency Test (Thurstone & Thurstone, 1962) The version employed here is that used and described by Benton (1968). The task requires subjects simply to say as many words beginning with the letter "F" as possible in a period of one minute, excluding proper nouns, numbers, and usages of the same word with a change in suffix. Subjects are then asked to do the same for the letters "A", and "S". This task arguably would be sensitive to "problems of starting" and of decreased

spontaniety, that is, to difficulties in shifting or modulating an ongoing inactive state in response to continued demand for new verbal productions.

Frontal lesions of either hemisphere have consistently been shown to result in significantly reduced production (raw number of responses) on this type of task relative to lesions in other brain areas, with left frontal impairment resulting in somewhat poorer performance than right (Miceli, Caltagirone, Gainotti, Masullo & Silveri, 1981; Perret, 1974). Bilateral lesions of the frontal lobes appear to depress scores even more (Benton, 1968). It may also be reasonably assumed that perseverative responses (i.e., repeated words) are reflective of frontal-lobe dysfunction, given that frontal-lobe patients produce a higher percentage of perseverative responses on a test considered to be a non-verbal analogue to this one (see Jones-Gotman & Milner, 1977).

Design Fluency Task. This test can be considered a non-verbal analogue of the speech fluency task (Jones-Gotman & Milner, 1977). It consists of two trials, in which the subjects are instructed to invent as many separate non-representational drawings as possible. The first trial is a "free" condition, in which subjects are told to invent drawings as they see fit, excluding drawings which can be recognized as objects, or which are

simply variations or rotations of a previous drawing. The
second trial is a "fixed" condition, having the limitation
that each drawing must consist of exactly four lines.

Like the speech fluency task, this test requires
consistent flexibility: the subject must repeatedly shift
to a new response. Two kinds of deficits have been
observed on this test. The first, a higher percentage of
perseverative or repetitive designs has been found in the
free condition for individuals with right frontal and
fronto-central lesions relative to controls, and in the
fixed condition for right frontal, right fronto-central,
and left frontal lesions (combined group) relative to
patients with lesions elsewhere. The second type of
deficit, a lower number of unique and acceptable designs
or "novel output" appears to have less specificity the
frontal areas. It has been observed in the free condition
for a combined right anterior lesion group (right frontal
plus right fronto-central plus right temporal) relative to
controls, and in the fixed condition for lesions in all
quadrants relative to controls. In this latter condition,
however, it does appear that right frontal and right
fronto-central groups exhibit the worst degree of impair-
ment (Jones-Gotman and Milner, 1977).

Converse Responding Task. In this task, the subject
is to respond conversely to a signal given by the

examiner. The examiner taps the table surface either once or twice. If once, the subject is to tap twice, and if twice, the subject is to tap once. To succeed on this task, the subject must repeatedly reprogram his response in relation to the examiner's varying signal. This reprogramming is made more complex in that the response is converse, thus demanding a cognitive shift with each response. Patients with <u>marked</u> frontal lobe lesions fare poorly on this type of task (Luria, 1966; Luria and Homskaya, 1964), falling into "mirror" reactions where the properties of their responses mimic those of the signal (i.e., when the examiner taps once, they tap once). It is unclear how individuals with less severe deficits perform on this task.

<u>Perseveration elicitation task</u>. The task requires subjects to draw simple geometric figures in a verbally commanded sequence as fast as they can. They must thus change their plan or program with each new command. Individuals with severe frontal lobe impairment show a tendency to continue some aspect of the immediately preceding design when a new design has been commanded (Luria, 1966; Luria and Homskaya, 1964). This tendency is evident whether commands are presented in written or printed form, or whether presented verbally (Lezak, 1983).

No information is available as to the effect of less than severe frontal impairment on this task.

WISC-R Mazes. This task requires subjects to continually alter directional movements of their pencil through the maze as additional information about the maze is perceived. This aspect of the task becomes especially salient in the more difficult mazes, in which it is more difficult to grasp the correct path in its entireity from the beginning. Evidence of difficulty in shifting behavior in response to new information might be found directly in the entry into blind alleys of the maze (a continuation of ongoing behavior), and indirectly in impaired performance time due to the errors and to slower shifting of behavior at critical junctures.

Patients following frontal lobotomy clearly exhibit impaired performance on the Porteus Maze test (Porteus, 1959; Tow, 1955), which is untimed and is scored only for errors defined as entry into incorrect paths of the maze. The Porteus Maze test also appears to be quite sensitive to the effects of brain damage in general (Klebanoff, Singer, & Wilensky, 1945). Lezak (1983) finds the WISC-R mazes a satisfactory subsitute for the Porteus Mazes.

Semmes Body Placing Test. The subject's task on this test is to point to the location on his or her body represented by numbers on a set of five schematic

diagrams, each of which presents both a front and back
view of a human figure drawn in outline. The lateral
reference points on the diagrams change, depending on
whether the point to be located is on the front or back
view of the human figure.

In order for the subject to maintain correct
left-right orientation while moving from points indicated
on the back view to those on the front view, and vice
versa, a cognitive shifting of frame of reference must be
repeatedly accomplished. Individuals with anterior lobe
lesions, particularly those of the left frontal region,
show impairment on this task relative to those with
posterior lesions. Left parietal lesions, however, also
appear to result in impaired performance, possible due to
problems with left-right discrimination (Kolb and Whishaw,
1980; Semmes, Weinstein, Ghent, & Teuber, 1963), or in
comprehension of how single parts relate to a whole
structure (De Renzi and Scotti, 1970); or to a more global
aphasic disorder (Lezak, 1983), rather than to difficul-
ties in shifting frame of reference per se.

Stroop Color-Word Test. This task derives from
Stroop's (1935) test in its numerous variations (Jensen &
Rohwer 1966; Dyer, 1971). There is no standard version of
the test with respect to materials, administration, and
scoring, but there is consistency as to the essential

nature of the task. In general, a list of color names (Stroop used red, yellow, blue, and green) is presented to the subject, each printed in ink of one of the named colors, but never in the color denoted by the word (e.g., the word "red" could be printed in blue, green, or yellow ink, but never in red). The subject is to name the color in which each word is printed under pressure of a timed trial. Performance time on this trial is usually compared to that of trials where the colors of a matrix of colored dots are named, and where color names are read from a list printed in black ink.

The test has been used in a variety of contexts, and there is uncertainty as to the processes it actually measures (Jensen & Rohwer, 1966). What is noteworthy for the current study is that the task presents a response competition situation, in which a color, demanding one verbal response (its name), and a word, demanding a different verbal response (its denotation), are presented simultaneously. There appears to be a natural tendency for the reading of the word to be a stronger response set than the naming of colors. Correct response requires a shift from the stronger "ongoing" response set to that of color naming. In order to make this shift, subjects must first inhibit the stronger, more automatically made

response set, and so the Stroop has been referred to as a test of "inhibitory control" (Kolb and Whishaw, 1980).

Normal subjects often find this task difficult, and are consistently slower in their ability to name the colors of the words than to read the words or name colors presented as color dots. Patients with frontal lobe lesions show significantly greater performance deficits from trials naming colors of dots (i.e., where there is no response competition) to response competition trials, relative to both controls and patients with lesions in other portions of the brain. There is also evidence of correlation between performance on this test and on the Speech Fluency Task described above (Perret, 1974).

Wisconsin Card Sorting Test (WCST) (Berg, 1948). The WCST uses a pack of sixty-four cards on which are printed either crosses, circles, squares, or triangles, varying in number (from one to four) and color (red, green, yellow, or blue). No two cards are identical. Subjects are required to place the cards one at a time under one of four stimulus cards: a red triangle, two green stars, three yellow crosses, and four blue circles. The examiner responds to each placement by indicating only whether it is "right" or "wrong". Correctness of place-ment is determined by the "sorting principle" in effect at the time (i.e., the cards must be sorted according to

either number, color, or form). This principle shifts
from one category to the next each time the subject makes
ten consecutive correct responses. No indication is given
to subjects that a shift has occurred, except that afford-
ed by the examiner's responses as to correctness.

The test thus requires the subject to make shifts
from established response strategies to alternate ones
(i.e., from sorting according to color, to sorting by
form, etc.) relative to variation in the signal provided
by the examiner. Frontal-lobe impaired individuals would
be expected to have specific difficulty making this type
of shift, and as a result would be expected to exhibit
reponses which appear to be "perseverative" in nature,
that is in which cards are sorted according to a principle
previously in effect even after the principle had changed.
Since it would also be difficult for these individuals to
obtain the ten consecutive correct responses required for
change of sorting principle, fewer changes in sorting
principle would also be expected in their test records.

The WCST one of the few tests generally accepted to
have specific sensitivity to frontal lobe brain lesions
(Heaton, 1981), and as a result has come into use as a
clinical neuropsychological instrument. Heaton (1981) has
performed a normative study for the WCST measures, and
group means are available for normals, brain damaged in

general, focal frontal, focal non-frontal, and diffuse groups.

Sorting tasks of various types have long been shown to be sensitive to frontal lobe impairment (Goldstein, 1944; Halstead, 1940; Nelson, 1976; Rylander, 1939; Weigl, 1941). There have been a number of studies demonstrating the WCST's sensitivity to Frontal Lobe dysfunction. Milner (1963; 1964) found that patients with superior dorsolateral frontal involvement were significantly impaired relative to patients with lesions elsewhere (including orbitofrontal) in terms of total number of errors, total number of sorting categories (i.e., shifts of sorting principle) achieved, and number of perseverative errors, but were no different from these control groups in terms of non-perseverative errors. Individuals with left hemisphere frontal involvement appeared to be more impaired than those with right hemi-sphere lesions.

Stuss, Benson, Kaplan, Weir, Naeser, Lieberman, & Ferril (1983) found that orbitofrontal leucotomized patients also suffered impairment in terms of number of categories achieved relative to patients with lesions elsewhere. Drewe (1974) found: 1) that patients with frontal lobe lesions completed fewer categories and made more perseverative errors than patients with lesions

elsewhere; 2) that lesions in the medial area of the frontal lobes may produce the greatest degree of such impairment; 3) that left-frontal patients show greater impairment than right-frontal in terms of total errors and number of categories, but both groups show equivalent impairment in terms of perseverative errors.

Robinson, Heaton, Lehman, & Stilson (1980) found frontal-impaired patients to be significantly more impaired than non-frontal in terms of the raw number of perseverative responses, with no lateralization effects. They also found the WCST perseverative error score to be a more sensitive predictor of frontal-lobe impairment than either the global impairment index of the Halstead-Reitan battery or any of the component measures from that battery. While frontal-lobe impaired patients were distinguishable from those with lesions elsewhere, they were not, however, significantly different from those with diffuse lesions. This is not too surprising, given that the frontal lobes constitute approximately forty percent of brain tissue. Nor is it surprising then, that the WCST was also found to be a good single index of brain damage, in that combined brain damaged groups were significantly worse than normals on all WCST indices.

In summary, there is good evidence that the WCST is specifically sensitive to frontal lobe impairment, at

least relative to impairment in other parts of the brain, with the perseverative response score and the category score being the most consistently sensitive measures. There is conflicting evidence regarding localization effects within the frontal lobes. The WCST does not appear to differentiate between frontal lesions and diffuse brain dysfunction. It does appear to be a good single measure of brain damage in general.

Review of Relevant Studies

Given the preceding discussion, a frontal lobe impaired subgroup within the conduct disorders should be recognizable as follows. First, if such a subgroup exists, it should exhibit the specific impairment associated with frontal or prefrontal lobe dysfunction: difficulties in making behavioral and mental shifts in response to changing internal and external circumstances. Second, these difficulties should occur supramodally, that is across a range of behavioral parameters, notably characterizing both the type of antisocial behavior exhibited, and performance on structured tasks (i.e., neuropsychological measures) requiring such behavioral and cognitive shifts. This range should be especially noteworthy if the underlying disability is of developmental nature, involving impaired maturation of a the frontal or prefrontal structures as a whole, as opposed to more

localized lesions. Finally, the above qualities should distinguish the subgroup from other conduct disordered individuals.

The above points in turn imply a set of questions that must be addressed in the design of any research investigating the existence of the subgroup in question. First, has the research utilized measures specifically tapping the expected difficulties in "shifting" to accomodate changing circumstances? Second, have these difficulties been assessed across both antisocial behavior and neuropsychological test performance parameters? And, has the range of measures been sufficient to establish supramodality of impairment to the degree expected if the underlying disability involves the whole of the prefrontal areas? Third, has the research contrasted individuals exhibiting the impairment in question with other conduct disordered individuals, thereby distinguishing a unique subgroup? In reviewing relevant studies, consideration of the degree to which these questions have been successfully addressed must precede interpretation of results.

Global neuropsychological deficits. There have been numerous studies investigating and generally confirming global neuropsychological impairment among antisocial individuals. These have limited direct bearing on the question of frontal lobe impairment, and are reviewed here

primarily because they establish precedent for viewing conduct disorders as neuropsychologically impaired. Most of these studies contrast global neuropsychological test performance of a globally defined experimental antisocial group with a group of normals, and do not distinguish subgroups within their antisocial populations.

Among those reporting positive findings is Fitzhugh (1973), who contrasted a group of court-referred delinquents with non-delinquent (but emotionally disturbed) clinic referrals, finding a significantly higher number of abnormal neuropsychological profiles among the delinquent group. Berman & Seigal (1976), Slavin (1978) and Yeudall, Fromm-Auch, & Davies (1982), also found significantly high incidences of abnormal neuropsychological profiles among delinquents, when compared to non-delinquent controls. Similarly high incidences of clinically abnormal neuropsychological profiles have also been found relative to normals among: persistent adult criminal offenders (Yeudall, 1978a); adult sex offenders, violent-aggressive criminals, adolescents with severe conduct disorders (Yeudall, 1978b; Yeudall & Fromm-Auch, 1979); violent adolescents in residential treatment (Spellacy, 1977); and juveniles with extensive criminal histories (Vorhees, 1981).

There have been a few studies comparing the global neuropsychological performance of groups defined by contrasting types of antisocial behavior. The typology of behavior used to define the groups in these studies, however, has reflected judicial and ethical concerns rather than behavioral differences corresponding to localization of brain function. Krynicki (1978), for example found that a group of behavior disordered patients with histories of multiple assaultive episodes exhibited global neuropsychological impairment relative to non-assaultive behavior disordered patients, who were indistinguishable from patients with a diagnosis of organic brain syndrome. A similar study, contrasting juvenile violent, non-violent and sexual offenders found no systematic group differences (Tarter, Hegedus, Alterman, & Katz-Garris, 1983). This contradictory result is expectable, given the lack of correspondence between the behavioral typology employed and those which might be suggested by differences in brain functioning.

There are, to my knowledge, no studies refuting the evidence of global neuropsychological impairment provided by the above described research. Lending additional support for this view are medically oriented (i.e., neurological) studies which report parallel findings of a high incidence of "soft signs" among antisocial groups,

for example, Karniski, Levine, Clark, Palfrey, & Metzler (1982), and Lewis, Shanok, Pincus, & Giammarino (1982). Even further validation is provided by a recent study demonstrating an association between neuropsychological deficits and neurological "soft signs" in serious delinquents (McManus, Brickman, Alessi, & Grapentine, 1985)

Frontal lobe deficits. Investigations into the incidence of deficits specifically implicating the frontal areas are limited both in number and scope. They are reviewed with reference to the relevant questions listed earlier in this section.

Pontius & Ruttiger (1976) compared 132 delinquent, normal, and "emotional problem" children using a blind administration of the Narratives Test, which purportedly classifies frontal lobe functioning according to four stages of maturity. While they found that significantly fewer delinquents achieved the highest stage of maturity using this measure (there were no differences between delinquents and "emotionals" nor between normals and "emotionals"), these results must be treated with caution. The Narratives Test essentially is a system for examining the written stories of individuals for evidence of ability to "switch the principle of action" as manifested by such switches in the actions described in the stories. Acceptance of face validity of this measure requires the

assumption that switches of action in a written story line are indicative of an individual's personal general ability to make such shifts. To my knowledge, there is no evidence supporting this assumption, nor is there empirical research (e.g., involving known brain-lesioned subjects) supporting an association with frontal lobe dysfunction. There is, in fact, some evidence that Narratives Test results do not correlate with a generally accepted measure of frontal lobe dysfunction, the perseverative response score of the Wisconsin Card Sorting Test (Stephaniv, 1985).

A second study, using a brief version of the Narratives Test, found 36 percent of a sample of young adult men charged with criminal acts demonstrated "specifically immature action behavior" associable with frontal lobe system dysfunction (Pontius & Yudowitz, 1980). Reservations regarding the Narratives Test also apply to this study, although a significant positive association between Narratives Test performance and results of Trail Making Test B, which may have some validity as a frontal lobe measure (Lezak, 1983), was also found. Neither this nor the previous study attempted to associate test performance with observable parameters of antisocial behavior. Neither study attempted a direct comparison of subgroups within the larger antisocial group.

A study by Sherrets (1980) compared performance of a group of 44 institutionalized delinquents to that of 18 juvenile psychiatric patients on the Luria Nebraska Neuropsychological Battery. A high incidence of brain dysfunction was found in both groups, with the psychiatric group evidencing a slightly greater degree and diffuseness of impairment. The localization scales of the battery, which are well correlated with dysfunction of specific cortical areas (Golden, Purisch, & Hammeke, 1979), indicated considerable frontal and parietal/occipital dysfunction within the delinquent group. There was no attempt to correlate this dysfunction with variations in type of antisocial behavior, nor was there an attempt to distinguish among subgroups of behavior disordered individuals, as all cases were compared to the Luria Nebraska norms. The Luria-Nebraska contains a fairly large number of test items which load on the frontal localization scale, and so it may be possible to assume supramodality of dysfunction given sufficient elevation of the scale. The study did not try to distinguish between the effects of the two cortical areas implicated (i.e., frontal and parietal/occipital).

The previously-cited study conducted by Yeudall, Fromm-Auch, & Davies (1982), in addition to finding a high incidence of abnormal neuropsychological profiles among

its sample of 99 delinquents, also found that the "specific pattern" of these deficits implicated anterior (i.e., frontal and/or temporal) brain dysfunction greater than posterior (occipital/parietal) in 99% of the sample. There was also a less striking finding of non-dominant hemispheric dysfunction greater than dominant in 60% of the sample.

Measures were the Halstead Reitan Battery plus 12 other neuropsychological measures, including at least one (a word fluency task) which has a strong association with frontal lobe functioning. There was, however, no direct test of differences between any groups (delinquent, normal, nor otherwise) on frontal lobe measures. Instead, the classification of localization was arrived at independently for each subject via clinical inference from the "specific pattern" of deficits. This method of classification did not, as indicated above, discriminate between frontal and temporal dysfunction, and succeeded in localizing the focus of dysfunction by brain quadrant only. There was no attempt to relate observed neuropsychological impairment to any qualities of antisocial behavior.

Two other previously cited studies by Yeudall and his colleagues, (Yeudall, 1978b; Yeudall & Fromm-Auch, 1979), found a pattern of deficits suggesting bilateral anterior (fronto-temporal) dysfunction in approximately 72

percent of persistent adult sex offenders and violent-aggressive criminals. These studies employed methods paralleling those of the study discussed immediately above, and consequently did not investigate behavioral as well as neuropsychological patterns, nor did they attempt to compare groups within the broader anti-social/conduct disorder population. Although a few appropriate frontal lobe measures were included in the test batteries, there was no direct test of relative performance on these measures.

A study conducted by Appellof (1986) compared the performance of 30 delinquents to that of 30 non-delinquents on a battery of 10 measures designed to assess prefrontal functioning. The battery included three tests included in some version in the current study: the Wisconsin Card Sorting Test, a Word Fluency Test, and the Porteus Mazes. No differences were found between groups on any of the measures. The author suggests that the absence of significant findings may stem from lack of attention to behavioral parameters of the delinquent group, which consisted of non-violent individuals. Obviously, this study did not discern among types of antisocial behavior within the group of concern, and any prefrontal or frontal effects could easily have been masked. The variety of measures utilized in this study

clearly was sufficient to establish supramodality of dysfunction, were it to occur.

Summary of relevant studies. The above research clearly confirms a high incidence of neuropsychological impairment among delinquents and criminals. It is likely that such impairment is at least partially characterized by a pattern of neuropsychological deficits associated with frontal lobe dysfunction, although these deficits are not well defined due both to methods of analysis employed and lack of specificity in the measures. There has been no attempt to clarify the relationship between these neuropsychological deficits and an observable behavioral syndrome. There has been no significant attempt to distinguish a frontal dysfunction subgroup from other conduct disorders, as studies either compare globally defined groups of delinquents to normals, or use procedures which do not directly test subgroup differences. The one study which could have profoundly addressed the question of supramodality of dysfunction produced no significant results, possibly due to lack of attention to behavioral differences within the experimental group.

The investigation attempts to improve upon previous studies by: 1) explicitly defining a subgroup for study according to behavioral symptomatology which might be associated with frontal lobe dysfunction; 2) by comparing

this experimental group to other conduct disorders rather than to normals; 3) by employing indices specifically sensitive to frontal lobe impairment in that they explicitly demand the type of flexible behavior which is mediated by the frontal lobes; 4) by utilizing a range and variety of tests sufficient to test a hypothesis of supramodality of dysfunction.

Specific Hypotheses

Two hypotheses are to be tested. The first is that conduct disordered individuals whose antisocial behavior can be characterized by difficulties in shifting response set when circumstances change will show impairment on neuropsychological measures associated with frontal lobe functioning relative to conduct disordered individuals who show no such behavioral difficulties. The second is that neuropsychological impairment will be supramodal, that is, will occur consistently across a variety of tasks associated with frontal lobe dysfunction.

METHOD

Subjects

Subjects were members of the student population of
a private secondary school specializing in the treatment
of adolescents with conduct problems. The school is
located in a Chicago suburb, and accepts students on
referral from many Chicago area school districts. Special
education funds provided by the referring school districts
on a per-student basis are the school's primary source of
revenue. Typically, students are referred when conduct
problems are so severe as to be beyond the scope of the
home school district's disciplinary and special education
resources. The students are thus often those who are
viewed as unmanageable, or as "lost causes" within their
home school setting. Many of the students have also
exhibited behavior problems away from school and are in
legal or family difficulty as a result.

The core of the school's program is a token economy
and level system, which is integrated into both academic
and social aspects of the curriculum. Detailed daily
token charts are maintained for each student, thus provid-
ing an ongoing record of both appropriate and problematic
behavior. Students are awarded tokens for increments of

appropriate behavior, and are "fined" tokens for inappropriate or disruptive behavior. Increasingly appropriate behavior, as indicated by accrual of tokens, is linked to increasingly higher "levels" of privilege. There is also a consistent "time out" procedure incorporated into the daily program. Generally, students are asked to remove themselves from the classroom to a designated time out area if disruptive behavior continues after several requests to stop or alter the behavior. If a student fails to remove him or herself upon request, then a forced removal (physical assistance by staff) follows. Removals are recorded on the daily token charts.

Students were selected for the study by a procedure designed to produce two groups with maximized contrast along dimensions of classroom behavior which might be associated with prefrontal symptomatology: difficulties in altering or switching ongoing behavior in response to changing circumstances (as described earlier in this paper). A description of the selection procedure follows.

1) Classroom teachers were individually consulted and asked to develop a list of students in their classrooms (grades 9 through 12 only) who most obviously exhibited the kind of difficulty in shifting or reprogramming ongoing behavior associated with pre-frontal dysfunction. The nature of the symptoms in question was

thoroughly reviewed with each teacher, and concrete examples were discussed until both teachers and examiner were convinced that the request was fully understood. Teachers were also asked to formulate a contrasting list of students who were conspicuous by the absence of the symptomatology in question.

2) Compilation of lists from all teachers produced a pool of 29 individuals thought to show frontal lobe symptoms (FLS), and 16 with no frontal symptoms (NFS). School records containing intelligence test results were reviewed, and all individuals functioning below the average range in terms of Wechsler equivalent IQ were eliminated from the pool.

3) Daily token charts of the remaining individuals were reviewed across an arbitrary six week period (30 school days) for evidences of classroom behavior associable with pre-frontal symptomatology. Two types of entries on the token chart were taken as the most likely to reflect pre-frontal symptomatology were it to occur. These were: a) repetitive sequences of two or more fines levied consecutively for the same problem behavior within a brief (5 minute) span of time - this was thought to reflect failure of an ongoing behavior to be modified by changed circumstances, even when unfavorable consequences were repeatedly made salient by the fines and accompanying

verbal information; b) removals from the classroom (as in
the above mentioned time out procedure) - within the
school's procedures, these resulted only after multiple
requests and opportunities to alter a behavior, and in the
face of continuing negative consequences, (i.e., fines).
The frequency of both types of entry were noted for each
remaining individual.

4) Students on the FLS list who had the highest
frequency of sequential fines and removals were, as much
as possible, matched for age, IQ and sex, with those on
the NFS list having the lowest frequency of sequential
fines and removals. For maximum contrast, the FLS
individuals with relatively lower frequency of sequential
fines and removals, and NFS individuals with high
frequency of these variables were eliminated.

5) Although the original goal for the study was to
have two groups of at least ten individuals each, the
above procedure resulted in two groups of only eight
students each. Rather than include additional students
marginally fitting these groupings, it was decided to
procede with the two groups of eight, comparable in terms
of IQ and sex, with maximum contrast on those indices in
the daily behavioral records most likely to reflect
pre-frontal symptomatology, and also congruent with
teacher observations and opinion.

Table 1 summarizes the characteristics of the groups across the relevant dimensions of contrast. The two groups were not significantly different (t tests, two-tailed) in terms of age, IQ, VIQ, and PIQ. The groups were significantly different at the .01 level in terms of repetitive fines per day, and removals per day. Furthermore, there was no overlap in the ranges of the two groups on the fines and removals variables. The two groups were also significantly different in terms of total fines per day, suggesting that there was a quantitative as well as qualitative difference in the antisocial behaviors exhibited by the two groups.

Although this quantitative difference cannot be considered a direct indicator of frontal lobe dysfunction, it can be viewed as a possible artifact of such organicity, since a decrease in overall performance is likely to accompany any frontal lobe-specific deficits. On the Wisconsin Card Sorting Test (Heaton, 1981), for example, the total number of errors also increases when perseverative errors (a specific frontal lobe symptom) increase. The higher number of total fines may also reflect a tendency for the selection process to pull for the overall worst-behaved and best-behaved individuals in the subject pool, or may be due to some unknown relationship between overall problematic behavior and frontal-lobe

TABLE 1

Summary of Descriptor Variables for the

Possible Frontal Lobe Symptoms (FLS) and

No Frontal Symptoms (NFS) Groups

Variable	FLS Group		NFS Group	
	Mean	SD	Mean	SD
Age	16.34	2.13	16.75	0.89
IQ	94.04	6.98	96.00	7.874
VIQ	91.07	9.02	95.13	8.46
PIQ	97.43	8.36	97.88	8.77
Repetitive fines/day	1.28	1.17	0.14	0.02
Removals/day	0.38	0.33	0.01	0.02
Total fines/day	4.74	3.48	0.71	0.49

Note: VIQ = Wechsler equivalent Verbal IQ

PIQ = Wechsler equivalent Performance IQ

linked problem behavior. In any case, this difference does not negate the qualitative differences between the groups. A finer look at this point, perhaps including an additional group which exhibited a high number of total fines, but few of the type expected for frontal lobe impaired individuals, might be a worthwhile future study.

Measures

Speech Fluency Task (Benton, 1968). Administration consisted of three trials, each preceded by verbal in-structions to say as many words as possible starting with each given letter in one minute, to exclude proper nouns, numbers, and repeats of the same word with a different suffix, and to begin upon the signal to "go". Prior to the initial trial, a practice trial, asking for three words starting with the letter "T", was given. This was fol-lowed by clarification as necessary. Scoring for the test follows Benton's method, consisting simply of a summation of all acceptable words produced over the three trials. Perseverative responses (repeats of a word) were also noted.

Lezak (1983) cautions that premorbid verbal skill level must be taken into account when evaluating this task: control subjects of low ability have a tendency to perform a little less well than brighter brain damaged patients in some research. A version of the test

appearing in Benton and Hamshes' Multilingual Aphasia Exam (1976) adjusts scores by adding points for lower educational levels and advanced age. However, since the current study involves groups equated for intelligence, such adjustment was deemed uneccessary.

Design Fluency. Administration and scoring followed Jones-Gotman & Milner (1977). Scoring was accomplished by an independent judge. All identifying marks on the drawing protocols were masked prior to scoring. Scores of primary concern for the study were the "percent perseverative" score, calculated for each condition by dividing the number of perseverative responses by the total number of drawings in that condition. "Novel output" scores as used by Jones-Gotman & Milner were not calculated because of their tendency to respond to lesions in non-frontal cortical areas.

Converse Responding (Luria & Homskaya, 1964). Administration was as follows: 1) the examiner explained that the subject was to knock twice if the examiner knocked once, and vice versa; 2) two practice trials using one and then two knocks were given, and corrective comments were provided; 3) a set of ten trials were given, with number of knocks in the following sequence: 1, 1, 2, 2, 1, 2, 1, 2, 2, 1; 4) the sequence of ten trials was repeated; 5) the examiner performed the knocks at an even,

but rapid pace, beginning each trial immediately following each subject response. Errors were noted as they occurred. The score was the total number of errors.

Perseveration Elicitation (Luria & Homskaya, 1964). Administration of this test began with presentation of a pencil and a blank, white, 8 1/2" x 11" sheet of paper. Subjects were asked to draw a circle, a cross, a triangle, and a square at their own rate to insure understanding of commands and sufficient motor ability. Subjects were then told to draw designs as commanded, as rapidly as possible. Four sets of commands were given. The order of geometric figures in each was as follows: a) trial 1 - circle, cross, circle, circle, circle; b) trial 2 - square, cross, circle, cross, cross, cross; c) trial 3 - triangle, square, triangle, square, square, square; d) trial 4 - cross, circle, circle, triangle, cross, circle, circle, triangle. Commands were given at intervals of 1 second.

The score was the number of perseverations, defined as the drawing of a previously commanded figure, or some partial aspect of the figure, to a subsequent command.

WISC-R Mazes. Administration was as per Wechsler's (1974) instructions. In addition to the Wechsler raw score, the raw number of entries into blind alleys was recorded.

Semmes Body Placing Test (Semmes, Weinstein, Ghent, & Teuber, 1963). Materials consisted of a five 11" x 14" cardboard plaques, each with diagrams showing full length views of both front and back view of a nude male figure, in heavy black outline. Each was marked with a series of numbers at various body parts. The diagrams were drawn by a professional artist, after those used by Semmes et al., 1963.

Subjects were instructed to touch parts of their own bodies in the order indicated by the numbers on the diagrams. The examiner also provided verbal cueing of the numbers as the task proceded. Plaques were presented each in turn, and additional explanations and encouragement were given as necessary. Scoring was for total number of incorrect responses. Self corrections were allowed if made without significant (e.g, about one second) delay.

Stroop Test (Stroop, 1935). Test materials were three 11" x 14" white cardboard plaques. The first of these, designated the (W) card had names of the colors red, blue, green, and brown (used instead of Stroop's original yellow because of better contrast with the white ground) printed upon it in black ink, arranged in a five column array, and occurring in random order. The words were hand lettered by a professional artist in easily readable block letters 1/4" high. The words were spaced

on center 1 1/2" apart horizontally, and 1" vertically. Each row was underlined with a solid black line approximately 1/24" thick, and was marked with a row number (in black ink) in the left margin to facilitate subjects' visual tracking of the words across the card.

A second card, the color (C) card, consisted of a a five by ten array of colored paper dots (red, blue, green, brown) glued to the plaque and spaced on center at intervals identical to those of the W card. Rows were numbered and underlined identically to the W card. Colors were in random sequence. The third card, the word-color (WC) card was identical to the W card, except that the words (arranged in the same sequence as the W card) were lettered in some other color (either red, blue, green, or brown) than that denoted by the word.

Administration consisted of four timed trials: 1) reading the W card; 2) naming the colors of the C card; 3) reading the denoted words on the WC card; 4) naming the colors of the WC card. Subjects were instructed to read the words (or name colors, as appropriate) as quickly as possible prior to presentation of each card. Cards were held by the examiner in a near upright position approximately 18" in front of the subjects and in their direct line of vision.

An inordinate number of scores have been extracted from the Stroop in its various uses (Jensen, 1965). Of primary concern for the current study, however, is the increased difficulty experienced in trial four due to the need to switch response set. The raw difference in color naming time between the WC card, and the C card (WC - C) was viewed as the most accurate reflection of this increased difficulty, following Perret (1974).

Wisconsin Card Sorting Test. Scoring and administration for this study were as per Heaton's revision (1981), except that only the first set of 64 cards were administered to each subject rather than the two complete sets, totalling 128. This was done due to time constraints on the administration. Total errors, number of categories achieved, and percent perseverative errors were considered to be the measures that most likely reflected the disability associated with frontal lobe dysfunction.

Procedure

Measures were administered as a battery in the order in which they are described above. Total time of administration for each subject was between three and four hours. Subjects were removed from their normal classroom, with teacher permission, for testing. They were allowed two five minute breaks at their own discretion between tasks during the battery.

Parental consent forms were required for participation in the study. Students were paid five dollars each for their participation, and were instructed verbally at the beginning of testing procedures that they could cease participation at any time, if they so elected. No students elected to cease participation.

Statistical analysis consisted of Mann-Whitney U tests for significant differences between groups on each of the dependent measures. Non-parametrics were viewed as appropriate for two reasons. First, although the two samples (NFS and FLS groups) were independent, the group selection process is likely to have violated the criteria of random sampling necessary for parametric tests. Second, much of the data is probably best thought of as at the ordinal level (e.g., the raw number of errors score on several measures), as opposed to the interval level required for parametrics (Seigel, 1956). Analysis was performed using the IBM-PC compatible version of the NPAR program of the SYSTAT statistical package (Systat, Inc., 1985).

RESULTS

The hypothesis of frontal lobe-associated neuropsychological deficits in the FLS group relative to the NFS group was tested by comparing the performance of the two groups across a total of 13 separate scores derived from the 8 measures. One-tailed Mann-Whitney $\underline{U}$ tests indicated significantly poorer performance of the FLS group relative to the NFS group ($\underline{p} \leq .05$) on 8 of the 13 scores. On 6 of the 8 tests, there was at least one score reaching significance in the expected direction. Mann-Whitney $\underline{U}$ test results are summarized in Table 2. Results of each test are reviewed below.

On the Wisconsin Card Sorting Test, group differences were significant in the expected direction for all three of the derived scores. The FLS group overall made more total errors, achieved fewer categories, and evidenced a higher number of perseverative responses than did the NFS group.

There were two separate scores derived from the WISC-R maze performance: raw number of errors and the "raw score" produced by the Wechsler scoring criteria. No significant difference between groups was observed in terms of raw number of errors, although the FLS group's

61

TABLE 2

One-tailed Mann-Whitney U Tests
on Dependent Measures for Possible Frontal
Symptoms (FLS) and No Frontal Symptoms (NFS) Groups

	FLS Group			NFS Group			
Variable	min	max	RS	min	max	RS	p
Wisconsin Card Sorting							
Total Errors	14.0	40.0	91.5	8.0	27.0	44.5	.01
Persever- ations	9.0	38.0	88.0	1.0	22.0	48.0	.02
Categories	0.0	3.0	42.5	2.0	4.0	93.5	.00
WISC-R Mazes							
Errors	0.0	18.0	77.0	1.0	11.0	59.0	.17
Raw Score	16.0	30.0	52.5	19.0	29.0	83.5	.05
Perseveration Elicitation							
Raw score	0.0	5.0	92.0	0.0	0.0	44.0	.00
Word Fluency							
Total responses	30.0	53.0	67.5	35.0	66.0	68.5	.48
Persever- ations	3.0	9.0	92.5	0.0	6.0	43.5	.00
Semmes Body Placing							
Raw errors	1.0	11.0	86.0	0.0	9.0	50.0	.03

Converse Responding

Raw errors   0.0   2.0   61.0   0.0   4.0   75.0   .22

Stroop

CW - C   16.5   29.0   80.0   16.3   31.6   56.0   .10

Design Fluency

% perseveration:

   Free con-
     dition   26.9   62.7   89.0   0.0   65.2   47.0   .01

   Fixed con-
     dition   9.1   57.7   81.5   0.0   62.5   54.5   .08

Note: min = minimum; max = maximum; RS = Rank Sum used in computation of Mann-Whitney $\underline{U}$

performance was in the expected direction (p = .17). Wechsler's raw score, which incorporates a penalty for time as well as error, was significantly lower (the expected direction) for the FLS group.

The FLS group was clearly impaired relative to the NFS group on the single score (raw number of errors) derived from the Perseveration Elicitation Task. In addition to the statistical difference in the expected direction, it is noteworthy that no perseverative responses were produced by any individual in the NFS group.

The first of the two Word Fluency scores, raw number of responses, did not differentiate between the groups. The FLS group did, however, produce a significantly higher percentage of perseverative responses, as predicted.

A single, raw error score was derived from the Semmes Body Placing test, and the FLS group produced significantly more errors than the NFS group. This result also was in keeping with hypothetical prediction.

On the Design Fluency test, the percent of perseverative responses made by the FLS group was higher relative to the NFS group in the free condition, but significance was not reached for the fixed condition. Group differences did, however, approach significance in the expected direction for the latter condition (p = .08).

No significant group differences were noted on the raw error score of the Converse Responding task, nor on the interference score (CW-C) of the Stroop Test. The latter score did approach significance in the expected direction (p = .10), however, with the FLS group showing a greater degree of interference relative to the NFS group. There were no other scores derived from these two tests.

The second hypothesis, that of consistency or supramodality across measures was tested by calculating the probability of obtaining results significant at the .05 level 8 out of 13 times by chance alone. Assuming that the measures used in this study are in fact all measures of the same phenomenon, the binomial probability for this occurrence is less than .0000 (Hays, 1980). Further support for the supramodality hypothesis arises when the variety of tests on which relative impairment was evidenced is considered. Impairment occurred respectively on tests emphasizing right hemisphere processes (Design Fluency), left hemisphere (i.e., verbal) processes (Word Fluency, visual-motor abilities and planning (WISC-R mazes, perseveration elicitation), integrated and/or abstract categorical thinking (Wisconsin Card Sorting), personal body orientation and awareness (Semmes Body Placing).

DISCUSSION

The major finding of this study is that
neuropsychological deficits on tests associated with
frontal or prefrontal dysfunction occur in a subgroup of
conduct disordered individuals who also show behavioral
evidence of such dysfunction, relative to conduct disor-
dered individuals showing no similar behavioral evidence.
Secondarily, the neuropsychological deficits occur across
a range of tests which vary in behavioral and cognitive
modality, and which are associated with different locales
within the frontal lobes, but which are nevertheless
similar in their demand upon an integral aspect of
prefrontal functioning: the ability to make cognitive or
behavioral shifts in response to changing demands and/or
circumstances.

It should be noted that even though significant
group differences were not observed on two of the eight
tests, one of these, the Stroop test, produced results
approaching significance in the expected direction. The
other, the Converse Responding Task, had previously only
been used with populations having severe frontal lobe
lesions, and so may simply have been inappropriate for the
population of the current study.

Overall, these findings strongly support the two specific hypotheses which were to be tested. The underlying purpose of this study, however, was to investigate the validity of conceptualizing a subgroup of conduct disorders as frontal lobe impaired, since confirmation of the existence of this subgroup is likely to have concrete implications for diagnosis and treatment. The following discussion attempts to address the question of how well the findings support the notion of a distinct frontal-lobe impaired subgroup within the conduct disorders. Implications for further research relative to diagnosis and treatment of such a subgroup are also addressed.

Of concern is the possibility that dysfunction in other areas of the brain, (i.e., non-frontal) may have affected the test performance of the FLS group. This plausible rival hypothesis deserves attention due to the previously noted sensitivity of several of the measures to lesions in a variety of brain locales. There are at least two factors which make this possibility rather implausible, however. First, the one measure which appears to come closest to being exclusively sensitive to dysfunction of the frontal lobes, the percent perseveration score of the Design Fluency Test, was dramatically worse for the FLS group than the NFS group. In previous research, this score was shown to differentiate between frontal-impaired

individuals and normals, but not between normals and any
other brain-damaged group (Jones-Gotman & Milner, 1977).

Second, the performance of the FLS group on the
various Wisconsin Card Sorting scores is considerably
worse than the norms for patients with non-frontal le-
sions, as published by Heaton (1981). On the Total Error
score, the mean of the FLS group was 56.0 (pro-rated,
since the current study administered 64 cards of the WCST,
while Heaton's means are for 128 cards), which is compara-
ble to the norm of 54.9 for patients with focal frontal
lesions. The Total Error score norm for focal non-frontal
patients is 37.6. The prorated FLS group mean on the
perseverative response score was 34.25, which is consider-
ably higher than the focal non-frontal norm of 28.0, yet
lower than the focal frontal norm of 48.8. It is likely
that the perseverative response mean of the FLS group
would be higher if the entire 128 cards were administered,
as many of the FLS subjects appeared to perseverate at an
increased ratio after initially achieving a category. In
terms of categories achieved, the pro-rated FLS mean of
3.0 is similar to the focal frontal norm of 3.1 and worse
than the focal non-frontal mean of 4.3.

Although it is doubtful that the performance of the
FLS group can be attributed to dysfunction in non-frontal
areas, it remains possible that global or diffuse

dysfunction, as opposed to specific and exclusive frontal impairment, is responsible for the observed deficits. This possibility is extremely difficult to discount. Since any diffuse impairment necessarily incorporates frontal impairment, the measures employed in this study must consequently be sensitive to its effects. For example, the published WCST norms are nearly identical on all scores for patients with known focal frontal lesions and those with diffuse damage (Heaton, 1981).

The only available argument against diffuse impairment being responsible for the observed deficits is that its presence requires the assumption of premorbid (or potential) IQ significantly higher for the FLS group than its observed mean IQ of 94.04. While dysfunction of or damage to specific brain areas, and in particular the frontal lobes, has virtually no effect upon overall performance on general IQ tests (Klebanoff, Singer, & Wilensky, 1945; Smith, 1960), diffuse impairment by its very nature necessarily implies a reduction of overall intellectual functioning. While this argument has a degree of merit on logical grounds, a premorbidly higher level of intelligence for the FLS group is quite possible. To fully resolve this question, research comparing the performance of a group similar to the FLS group across measures specifically sensitive to other brain areas, as

well as the frontal lobes, might be conducted. IQ scores
obtained prior to the onset of the frontal-like symptoms
would also be useful in that an earlier higher IQ would
likely reflect a diffuse organic process.

Regardless of whether the observed frontal lobe
symptoms occur uniquely or within the context of more
diffuse impairment, the results of the study are suffi-
ciently conclusive to warrant development of experimental
treatments specifically aimed at remediation or rehabili-
tation.   Such treatments might take the form of
remedial training similar to that employed in the treat-
ment of developmental disablities, or to the kind of
procedures more recently coming into vogue under the
rubric of "behavioral neuropsychlogy" (Blanton & Gouvier,
1986; Puente & Hoston, 1986). Pontius (1972) has suggest-
ed that "cognitive training", consisting of practice with
tasks requiring appropriate types of behavioral and/or
mental shifts, such as those employed as test instruments
in this study, might also be effective.   Practice with
such tasks might allow individuals to develop alternative
coping strategies, based on cognitive processes which do
not lean heavily on frontal lobe functioning. Cognitive
strategies and coping skills developed in this way might
then be extended to role-playing more realistic situations
where a need for mental and /or behavioral shifts is

likely to be manifested, and ultimately to in vivo train-
ing. Screening procedures, which could lead to early
identification and preventative treatment, would be an
essential part of any programmatic treatment effort.

As treatments are developed and tested, the dis-
tinction between diffuse and focal frontal impairment may
ultimately prove superfluous, since rehabilitative efforts
targeting frontal-lobe cognitive deficits are likely to
follow a similar paradigm for either type of impairment.
Studies investigating the relative efficacy of treatment
might in fact serve to further investigate this issue by
attending to differential response to treatments among
those individuals exhibiting frontal lobe symptoms.

Prior to development of treatment for this
subgroup of conduct disorders, it would also be helpful to
have an efficient means of screening and/or diagnosing
individuals with possible frontal-lobe impairment. The
consistency of results of this study across its several
measures suggests that the test battery as a whole or in
some part might be developed into an extremely accurate
diagnostic tool.

A closer look at the pattern of test results,
however, suggests that this may be unneccessary, as the
Wisconsin Card Sorting Test appears to have strong poten-
tial for use as a screening device when all three

pertinent scores (total errors, categories achieved, and perseverative responses) are considered. In the current study, only one individual of the eight in the NFS group had performed above the norms for Heaton's focal frontal group on as many as one of the WCST scores. Only two individuals in the FLS group achieved less than one WCST score below these norms. Thus, when a cut-off criteria of any one of the three scores above focal frontal norms is applied to the population of the current study, an overall correct classification rate in excess of 81% is achieved. This incorporates a false positive rate of 6.25% (1 in 16), and a false negative rate of 12.5% (2 in 16).

These are, by any estimation, very good rates of classification, and further investigation of the WCST's utility as a screening instrument, particularly in conjunction with behavioral observations like those used in this study's group selection process, seems well worth while. For the time being, screening for research purposes could be accomplished by using a cutting score of two or three scores above the focal frontal norms, as this would minimize the number of false positives. For treatment related screening, a cutting score of one or possibly two seems more suitable, as this minimizes false negatives. Table 3 summarizes the distribution of cases

TABLE 3

Number of Wisconsin Card Sorting Scores Above

Focal Frontal Norms by Group

# of Scores Above Norm	FLS Group	NFS Group
0	2	7
1	1	1
2	4	0
3	1	0

Note: FLS = Possible Frontal Lobe Symptoms; NFS = No
Frontal Lobe Symptoms.

in each of the two experimental groups across the number
of WCST scores above the focal frontal norms.

The perseveration elicitation task employed in this
study may also prove to have utility as a screening
device, since none of the subjects in the NFS group showed
any evidence of impaired performance on it - a 100%
correct classification rate before shrinkage. The task
has had only limited use, however, and there is no
normative data. There is also a fairly strong subjective
element to the scoring procedure. Pending further re-
search, the task should probably only be employed as a
screening device in conjunction with other instruments.
For the time being, it would probably make a good validity
check on the WCST.

The findings of this study say nothing about the
possiblity of other neuropsychologically defined subgroups
within the conduct disorders. Certainly it would be
feasible to conduct studies, parallelling this one, which
would attempt to find convergence between behavior pat-
terns correlating with dysfunction of other cortical areas
and neuropsychological measures. The positive results of
the present study should serve to encourage this type of
research.

The study also says nothing regarding the etiology
of the observed impairment. The supramodality of

dysfunction observed is consistent with Pontius' (1972)
hypothesis of developmental delay of frontal lobe func-
tion, but traumatic damage or perhaps nutritional deficits
might produce similar results. Also remaining unanswered
is the rather important question of whether the observed
frontal lobe impairment is in fact a causative factor in
development of a diagnosable conduct disorder. There is no
way to adequately address this question without employing
a prospective research design, identifying individuals
with frontal lobe symptoms at an early age and determining
how many of these later develop conduct disorders.

One study using a prospective design was conducted
by Spreen (1981) with decidedly negative results, finding
no association between brain damage and delinquency nor
between "learning disabilities" and delinquency. It may
well be that the presence of brain dysfunction does not
significantly increase the likelihood of behavior prob-
lems. This does not imply however, that the diagnosis of
brain dysfunction in conduct disorders is spurious. It is
more likely, as some researchers have recently suggested,
that the diagnosis of conduct disorder itself has limited
utility (Lewis, Lewis, Unger, & Goldman, 1984), reflecting
a tendency to classify according to the non-criterial, but
extremely salient common symptom of aggression or vio-
lence. As these authors note, aggression or violence is a

"non-specific symptom" and may reflect any number of psychiatric conditions, including psychosis, manic states, borderline retardation, neurological impairment and learning disabilities. As additional data sheds light upon the underlying causes of conduct and behavior problems, and specific treatments are developed to deal with each of these, the diagnosis of conduct disorder may give way to more specific syndromes based on varying genotypes or causes.

Conduct disorders are a serious problem for both
the mental health community and society as a whole due to
their high prevalence, poor prognosis, and pessimistic
treatment outlook. The lack of treatment success with
conduct disorders may reflect the existence of several
distinct subgroups, varying in etiology and in response to
treatment, within the diagnostic classification. One
possible subgroup might be defined in terms of symptoms of
frontal lobe dysfunction. Clear identification of such a
subgroup would lead to the development of more specific
and effective diagnostic and treatment procedures. This
study proposes to investigate the validity of conceptual-
izing a subgroup of the conduct disorders as frontal lobe
impaired by testing for convergence between behavioral and
neuropsychological indicators of frontal dysfunction.

The roots of the study are in the Minimal Brain
Dysfunction (MBD) Research of the 1960's and early 1970's,
which attempted to link a wide range of childhood behav-
ioral and learning problems to a general underlying
organic problem. Although no evidence for a global
symptom complex or syndrome associated with signs of
impaired central nervous system functioning was found, a

number of suggestions for further research were generated. Among the more salient of these was that of delineating subsets of problematic children according to symptoms grouped on the basis of localization of brain dysfunction.

The current study was designed to delineate a subset according to symptoms grouped on the basis of frontal lobe dysfunction, perhaps the most integral of which is a difficulty in making appropriate mental or behavioral shifts in response to changing internal and/or external demands. This symptom is tends to be supramodal, and should thus be observable a wide range of tasks and behaviors, including neuropsychological measures. It should also characterize the antisocial behavior of some individuals, who may come into conflict with society because they are unable to "reprogram" or shift their actions appropriately, even when the consequences are quite negative.

Although previous research confirms a high incidence of neuropsychological impairment among conduct disorders, and further suggests that such impairment is at least partially characterized by a pattern implicating frontal lobe dysfunction, the relationship between these neuropsycholgocial deficits and an observable behavioral syndrome remains unclarified. Furthermore, a frontal dysfunction subgroup has yet to be distinguished from

other conduct disorders. Most relevant studies have either compared globally defined groups of delinquents to normals, or used procedures which fail to test subgroup differences. The current study specifically tested the hypothesis that conduct disordered individuals showing difficulties in shifting response set when circumstances change (the integral frontal impairment symptom) will also show impairment on neuropsychological measures associated with frontal lobe functioning when compared to conduct disordered individuals who show no such behavior difficulties. Secondarily, the hypothesis of supramodality of dysfunction was tested.

Two groups of eight students each in grades 9 through 12 were selected from the population of a school for conduct disordered students. The group selection procedure maximized contrast between the two groups on behavioral dimensions characterizing frontal lobe symptomatology. Teacher report and daily individual behavior charts were used for this purpose. The groups were, as best possible, matched for IQ, age, and sex. All subjects were in the average range of intelligence (Wechsler IQ: 85 - 115).

A battery of eight tests was administered, each arguably consisting of a task requiring mental or behavioral shifts in response to varying internal and/or

environmental signals. For inclusion in the battery, tests were required to have documented sensitivity to frontal lobe dysfunction. Tests incorporating a wide variety of specific task were included in the battery, so as to demonstrate supramodality of dysfunction, should it occur. Thirteen separate measures were extracted from the 8 tests.

Data analysis resulted in significant differences ($\underline{p}$ < .05, one-tailed) between the two groups in the expected direction on 8 of the 13 separate measures. Evidence of impairment for the group exhibiting behavioral symptoms of frontal lobe dysfunction was observable, relative to the contrasted group, on 6 of the eight separate tests. These results generally confirm the two experimental hypotheses, and are unlikely to have been due to focal lesions in non-frontal portions of the cortex. It is impossible, however, to rule out the possibility that the observed frontal lobe deficits may have occurred in the context of global or diffuse brain dysfunction. This may not be a useful distinction, however, if the majority of the subgroup proves to respond to similar rehabilitative treatment. Further research will be necessary to clear up this matter, as well as to determine the etiology and course of impairment, and ultimately, its specific role in conduct problems. Of secondary interest

for future studies is the finding that the Wisconsin Card Sorting Test alone may prove to be an effective screening device for frontal lobe dysfunction in this population.

REFERENCES

American Psychatric Association (1980). <u>Diagnostic and Statistical Manual of Mental Disorders</u>, edition 3, Washington, D. C.: American Psychiatric Association.

Appellof, E. S. (1986). Prefrontal functions in juvenile delinquents. <u>Dissertation Abstracts International</u>, 46, 3206-B.

Benton, A. L. (1968). Differential behavioral effects in frontal lobe disease. <u>Neuropsychologia</u>, 6, 53-60.

Benton, A. L., & Hamsher, K. DeS. (1976). <u>Multilingual Aphasia Examination</u>. Iowa City: University of Iowa, (<u>Manual</u>, revised 1978).

Berg, E. A. (1948). A simple objective technique for measuring flexibility in thinking. <u>Journal of General Psychology</u>, 39, 15-22.

Berman, A., & Seigal, A. (1976). Adaptive and learning skills in juvenile delinquents: A neuropsychological analysis. <u>Journal of Learning Disabilities</u>, 9, 51-58.

Blanton, P. D., & Gouvier, W. D. (1986). Cognitive retraining therapies for neurologically impaired patients. <u>The Behavior Therapist</u>, 9(3), 47-50.

Chess, S. (1972). Neurological dysfunction and childhood behavioral pathology. <u>Journal of Autism and Childhood Schizophrenia</u>, 2(3), 299-311.

Clements, S. (1966). <u>Minimal brain dysfunction in children</u>. NINDB Monograph No. 3. Washington, D. C.: U. S. Public Health Service.

Coleman, J., Butcher, J., & Carson, R. (1980). <u>Abnormal Psychology and Modern Life</u> (6th ed). Glenview, Illinois: Scott, Foresman & Co.

De Renzi, E., & Scotti, C. (1970). Autotopagnosia. Fiction or reality? <u>American Medical Association Archives of Neurology</u>, 23, 221-227.

Drewe, E. A. (1974). The effect of type and area of brain lesion on Wisconsin Card Sorting Test performance.

Cortex, 10, 159-170.

Dyer, F. N. (1971). A comparison of chromatic and achromatic versions of the Stroop color-word test. Psychonomic Science, 22, 235-237.

Feldhusen, J., Benning, J., & Thurston, J. (1972). Prediction of delinquency, adjustment and academic achievement over a five-year period with the Kravaraceus Delinquency Proneness Scale. The Journal of Educational Research, 65(8), 375-381.

Fitzhugh, K. (1973). Some neurological features of delinquent subjects. Perceptual and Motor Skills, 36, 494.

Gesell, A. & Armatrauda, C. (1941). Developmental Diagnosis. New York: Hoeber.

Golden, Purisch, & Hammeke (1980). Luria-Nebraska Neuropsychological Battery: Manual. Los Angeles: Western Psychological Services.

Goldstein, K. (1944). Mental changes due to frontal lobe damage. Journal of Psychology, 17, 187-208.

Halstead, W. C. (1940). Preliminary analysis of grouping behavior in patients with cerebral injury by method of equivalent and non-equivalent stimuli. American Journal of Physiology, 96, 1263-1294.

Hays, W. (1980). Statistics, (3d ed). New York: Holt, Rinehart & Winston.

Heaton, R. K. (1981). Wisconsin Card Sorting Test Manual. Odessa, Florida: Psychological Assessment Resources, Inc.

Jenkins, R. (1964). Diagnoses, dynamics and treatment in child psychiatry. In R. Jenkins & J. Cole (Eds.), Research Report No. 18. Washington, D. C.: American Psychiatric Association.

Jensen, A. (1965). Scoring the Stroop test. Acta Psychologica, 24, 398-408.

Jensen, A. & Rohwer, W. (1966). The Stroop color-word test: A review. Acta Psychologica, 25, 36-93.

Jones-Gotman, M. & Milner, B. (1977). Design fluency, the invention of nonsense drawings after focal cortical lesions. <u>Neuropsychologia</u>, 15, 653-674.

Karniski,, W. M., Levine, M. D., Clarke, S., Palfrey, J. S., Meltzer, L. J. (1982). A study of neurodevelopmental findings in early adolescent delinquents. <u>Journal of Adolescent Health Care</u>, 3, 151-159.

Klebanoff, S. G., Singer, J. L., & Wilensky, H. (1945). Psychological consequences of brain lesions and ablations. <u>Psychological Bulletin</u>, 42, 585-623.

Kolb, B. & Milner, B. (1981). Performance of complex arm and facial movements after focal brain lesions. <u>Neuropsychologia</u>, 19(4), 491-503.

Kolb, B. & Whishaw, I. (1980). <u>Fundamentals of Human Neuropsychology</u>. San Francisco: W. H. Freeman and Company.

Krynicki, V. (1978). Cerebral dysfunction in repetitively assaultive adolescents. <u>Journal of Nervous and Mental Disorders</u>, 166, 59-67.

Lewis, D. & Balla, D. (1976). <u>Delinquency and Psychopathology</u>. New York: Grune & Stratton.

Lewis, D. Shanok, S., Pincus, J., & Giammarino, M. (1982). The medical assessment of seriously delinquent boys: A comparison of pediatric, psychiatric, neurologic, and hospital record data. <u>Journal of Adolescent Health Care</u>, 3(3), 160-164.

Lewis, D., Lewis, M., Unger, L., & Goldman, C. (1984). Conduct disorder and its synonyms: Diagnoses of dubious validity and usefulness. <u>The American Journal of Psychiatry</u>, 141, 514-519.

Lezak, M. (1983). <u>Neuropsychological Assessment</u>, 2d edition. New York: Oxford University Press.

Lombroso, C. (1910). <u>The Man of Genius</u>. New York: Charles Scribner's Sons.

Luria, A. R. (1966). <u>Higher Cortical Functions in Man</u>. New York: Basic Books.

Luria, A. R. & Homskaya, E. D. (1964). Disturbances in the regulative role of speech with frontal lobe lesions. In J. M. Warren and K. Akert, (Eds.), The Frontal Granular Cortex and Behavior. New York: McGraw-Hill.

McFie, J. (1975). Assessment of organic intellectual impairment. London: Academic Press.

McManus, M., Brickman, A., Alessi, N., Grapentine, W. (1985). Neurological dysfunction in serious delinquents. Journal of the American Academy of Child Psychiatry, 24(4), 481-486.

Meeks, H. E. (1980). Conduct disorders. In A. M. Freedman, H. I. Kaplan, & B. J. Sadock (Eds.), Comprehensive Textbook of Psychiatry. Baltimore: Williams and Wilkins.

Miceli, G., Caltagirone, G., Gainotti, G., Masullo, C., & Silveri, M. C. (1981). Neuropsychological correlates of localized cerebral lesions in non-aphasic brain-damaged patients. Journal of Clinical Neuropsychology, 3, 53-63.

Milner, B. (1963). Effects of different brain lesions on card sorting. Archives of Neurology, 9, 90-100.

Milner, B. (1964). Some effects of frontal lobectomy in man. In J. M. Warren and K. Akert, Eds., The Frontal Granular Cortex and Behavior. New York: McGraw-Hill.

Nelson, H. (1976). A modified card sorting test sensitive to frontal lobe defects. Cortex, 12, 313-324.

Pasamanick, B., & Knobloch, H. (1960). Brain damage and reproductive casualty. American Journal of Orthopsychiatry, 30, 298-305.

Perret, E. (1974). The left frontal lobe of man and the suppression of habitual responses in verbal categorical behavior. Neuropsychologia, 12, 323-330.

Peterson, D., Quay, H., & Anderson, A. (1959). Extending the construct validity of a socialized scale. Journal of Consulting Psychology, 23, 182.

Peterson, D., Quay, H., & Cameron, G. (1959). Personality and background factors in juvenile delinquency as inferred from questionnaire responses. Journal of Consulting Psychology, 23, 395-399.

Pincus, J., & Tucker, G., (1978). Behavioral Neurology. New York: Oxford University Press.

Pond, D. A. (1967). Behavior disorders in brain-damaged children. In Modern Trends in Neurology, 4, 125-134.

Pontius, A. (1972). Neurological aspects in some types of delinquency. Adolescence, 7(27), 289-308.

Pontius, A. (1973). A Conceptual model of Minimal Brain Dysfunction. (General Discussion, Proceedings of Minimal Brain Dysfunction Conference, 1972), Annals of the New York Academy of Science, 205, 61-63.

Pontius, A., & Ruttiger, K. (1976). Frontal lobe system maturational lag in juvenile delinquents shown in narratives test. Adolescence, 11, 509-518.

Pontius, A., & Yudowitz, B. (1980). Frontal lobe system dysfunction in some criminal actions as shown in the Narratives Test. Journal of Nervous and Mental Disease, 168, 111-117.

Porteus, S. D. (1959). The Maze Test and Clinical Psychology. Palo Alto, California: Pacific Books, 52-82.

Puente, A. E., & Hoston, A. M. (1986). Behavior Therapy and Clinical Neuropsychology: Behavioral Neuropsychology. The Behavior Therapist, 9(2), 30-31.

Robins, L. (1966). Deviant Children Grown Up. Baltimore: Williams & Wilkins.

Robinson, A., Heaton, R., Lehman, R., & Stilson, D. (1980). The utility of the Wisconsin Card Sorting Test in detecting and localizing frontal lobe lesions. Journal of Consulting and Clinical Psychology, 48(5), 605-614.

Routh, D. & Roberts, R. (1972). Minimal brain dysfunction in children: Failure to find evidence for a behavioral syndrome. Psychological Reports, 31, 307-314.

Rutter, M. (1970). Psycho-social disorders in childhood and their outcome in adult life. Journal of the Royal College of Physicians, London, 4, 211-218.

Rutter, M. (1982). Syndromes attributed to minimal brain dysfunction in childhood. American Journal of Psychiatry, 139(1), 21-33.

Rutter, M., Graham, P., & Yule, W. (1970). A Neuropsychiatric Study in Childhood. Clinics in Developmental Medicine, Nos. 35/36, London: Heinemann.

Rylander, G. (1939). Personality Changes After Operations on the Frontal Lobes. London: Oxford University Press.

Saklofske, D. (1977). Personality and behavior problems of schoolboys. Psychological Reports, 41, 445-446.

Saklofske, D., McKerracher, D., & Eysenck, S. (1978). Eysenck's theory of criminality: A scale of criminal propensity as a measure of antisocial behavior. Psychological Reports, 43, 683-686.

Schulman, J., Kaspar, J., & Throne, F. (1965). Brain Damage and Behavior: A Clinical-experimental Study. Springfield, Ill.: Charles C. Thomas.

Seigel, S. (1956). Nonparametric Statistics for the Behavioral Sciences. New York: McGraw-Hill.

Semmes, J., Weinstein, S., Ghent, L., & Teuber, H-L. (1963). Correlates of impaired orientation in personal and extrapersonal space. Brain, 86, 747-772.

Sherrets, S. D. (1980). Behavior disorders in adolescents: Neuropsychological and behavioral correlates. Dissertation Abstracts International, 41, 2780B-2781B.

Slavin, S. (1978). Information processing defects in delinquents. In L. J. Hippchen, Ed., Ecologic-Biochemical Approaches to Treatment of Delinquents and Criminals. New York: Van Nostrand Reinhold, 75-104.

Smith, A. (1960). Changes in Porteus Maze scores of brain operated schizophrenics after an eight-year interval. Journal of Mental Science, 106, 967-978.

Spellacy, F. (1977). Neuropsychological differences between violent and nonviolent adolescents. Journal of Clinical Psychology, 33, 966-969.

Spreen, O. (1981). The relationship between learning disability, neurological impairment, and delinquency: Results of a follow-up study. Journal of Nervous and Mental Disease, 169, 791-799.

Stephaniv, W. (1985). Verification of the narratives test with perseveration responses on the Wisconsin Card Sorting and Category tests. Dissertation Abstracts International, 40, 2637-A.

Strauss, A. & Lehtinen, L. (1947). Psychopathology and education of the brain-injured child. New York: Grune and Stratton.

Stroop, J. (1935). Studies of interference in serial verbal reactions. Journal of Experimental Psychology, 18, 643-662.

Stuss, D., Benson, D., Kaplan, E., Weir, W., Naeser, M., Lieberman, I., & Ferril, D. (1983). The involvement of orbitofrontal cerebrum in cognitive tasks. Neuropsychologia, 21(3), 235-248.

Systat, Inc. (1985). SYSYAT. The System for Statistics, Evanston, Illinois: Systat, Inc.

Tarter, R., Hegedus, B., Alterman, A., & Katz-Garris, L. (1983). Cognitive capacities of juvenile violent, nonviolent, and sexual offenders. The Journal of Nervous and Mental Disease, 171, 564-567.

Thurstone, L. L. & Thurstone, T. G. (1962). Primary Mental Abilities (Rev.). Chicago: Science Research Associates.

Tow, P. M. (1955). Personality Changes Following Frontal Leucotomy. London: Oxford University Press.

Tucker, G. J. & Pincus, J. H. (1980). Child, adolescent, and adult antisocial and dysocial behavior. In A. M. Freedman, H. I. Kaplan, & B. J. Sadock (Eds.) Comprehensive Textbook of Psychiatry. Baltimore: Williams and Wilkins.

Voorhees, J. (1981). Neuropsychological differences between juvenile delinquents and functional adolescents: A preliminary study. Adolescence, 16, 57-66.

Wechsler, D. (1974). Manual for the Wechsler Intelligence Scale for Children - Revised. New York: The Psychological Corporation.

Weigl, E. (1941). On the psychology of so-called process-es of abstraction. <u>Journal of Abnormal and Social Psychology</u>, 36, 3-33.

Wender, P., and Eisenberg, L. (1974). Minimal brain dysfunction in children. In S. Arieti (Ed.), <u>American Handbook of Psychiatry</u> (2nd edition), Vol. ii. New York: Basic Books, Inc.

Werry, J. S. (1979). Organic factors. In H. C. Quay & J. S. Werry (Eds.), <u>Psychopathological Disorders of Childhood</u> (2nd edition). New York: John Wiley & Sons.

Yeudall, L. (1978a). Neuropsychological correlates of criminal psychopathy. I. Differential diagnosis. In L. Belveau, G. Canepa, & D. Szabo, Eds., <u>Human Aggression and Dangerousness</u>. Pinel Institute, Montreal, Quebec, Canada.

Yeudall, L. (1978b). The neuropsychology of aggression. <u>Clarence M. Hincks Memorial Lectures: Psychobiological Approaches to Aggression in Mental Illness and Mental Retardation</u>. London, Ontario, Canada.

Yeudall, L. & Fromm-Auch, D. (1979). Neuropsychological impairments in various psychopathological populations. In Gruzelier, J., and Flor-Henry, P., Eds., <u>Hemispheric Assymetries of Function in Psychopathology</u>. Elsevier, Amsterdam.

Yeudall, L., Fromm-Auch, D., & Davies, P. (1982). Neuropsychological impairment of persistent delinquency. <u>Journal of Nervous and Mental Disease</u>, 170(5), 257-265.

APPROVAL SHEET

The dissertation submitted by Robin C. Shear has been read and approved by the following committee:

Alan DeWolfe, Ph.D., Director
Professor, Department of Psychology
Loyola University

John Shack, Ph.D.
Associate Professor, Department of Psychology
Loyola University

Jill Reich, Ph.D.
Associate Professor, Department of Psychology,
and Associate Dean of The Graduate School
Loyola University

The final copies have been examined by the director of the dissertation and the signature which appears below verifies the fact that any necessary changes have been incorporated and that the dissertation is now given final approval by the Committee with reference to the content and form.

The dissertation is therefore accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

7/2/87
_____
Date

_____
Director's Signature